ENCLOSURE 9

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
ENVIRONMENTAL RESEARCH LABORATORY — DULUTH
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March 25, 1992

MEMORANDUM

SUBJECT: Columbia River Dioxin TMDL and Bald Eagles: Rebuttal of Ian Nisbet Declaration of 2/28/92

FROM: Steven Bradbury, Chief
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TO: Richard Albright, Chief
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Office of Water Planning
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Listed below are comments directed to the issues raised by Ian Nisbet regarding the dioxin risk analysis I provided in my declaration of February 12, 1992. I have referenced my rebuttal points with the Paragraph numbers in his declaration that is dated February 28, 1992.

Paragraphs 3,4,6,7,8,9,10. My February 12 declaration addressed dioxin effects on bald eagles. The fact that I did not controvert statements in Ian Nisbet's declarations unrelated to dioxin effects on bald eagles, or to issues only marginally related to the dioxin assessment in my declaration, should not necessarily be interpreted as concurrence with his assertions.

Paragraph 4. As a point of clarification I would like to remind you that the Office of Research and Development (ORD) within the U.S. EPA is currently reassessing the health and environmental risks of dioxin. Part of the reassessment involves additional research. The interim results of this reassessment are planned for release this spring or early summer and a final assessment is expected in the Fall of 1993. The reassessment effort will assist U.S. EPA in determining whether or not to modify recommendations in the current dioxin water quality criteria document.

Paragraph 5. Dr. Nisbet seems to be incorrectly implying the dioxin BAF of 90,000 used in the eagle risk assessment is also appropriate for human health risk assessments. This BAF is based on whole-fish at a 9% lipid content and is not appropriate for use in assessing human health risks.

Paragraphs 6, 7, 8, 9, 10, 13, 24, 23, 25c, 25d. My declaration was not designed to address whether or not eagles are currently at risk due to dioxin exposure; however, note that the study by Garrett et al. (1988) implicates DDE as the most likely chemical contaminant putting eagles at risk. Rather my assessment establishes that a future ambient aqueous dioxin concentration of 0.013 ppq can be reasonably assumed to protect eagles from drinking water and dietary intake of the compound. It is reasonable to contend that reducing the dioxin load from pulp and paper mills by 95%, by implementing the TMDL, would put the eagles at significantly less risk since their exposure to the chemical would be reduced. The absolute magnitude of the current dioxin-specific risk has not been quantified by anyone, including Dr. Nisbet.

Dr. Nisbet can, at best, claim to have provided a potential hazard identification, by citing several field studies and some laboratory studies, that a mixture of dioxin like compounds, as well as DDE and other organochlorine insecticides, may be placing the eagles at risk. He does not, however, provide a hazard assessment and an exposure assessment (as best as I can ascertain his exposure analysis in Paragraph 24 is based on very preliminary and incomplete information) that quantifies the risk to bald eagles of exposure to dioxin and the other chemicals. He is using a semi-quantitative analysis to identify a potential current hazard. His declaration in the cited paragraphs does not in any way refute my assessment that future dioxin residue levels in fish associated with an ambient aqueous dioxin concentration of 0.013 ppq will be protective for eagles. To refute the appropriateness of the 0.013 ppq dioxin value requires a quantitative, not a qualitative, analysis of future risk, which Dr. Nisbet never provides.

With regard to the mixture issue, it is my opinion that the TEF approach for wildlife risk assessments is very difficult to defend at this time. I would certainly not want to defend such an approach. Conceptually the TEF approach is reasonable, but it is insufficiently developed, in my opinion, to be used to establish wildlife criteria for issuing permits. The TEFs for dioxin and furans that are currently specified by the Agency were not meant to apply to wildlife. Analysis of the data was focused on studies relevant to human carcinogenicity as the primary endpoint of concern. Development of a set of TEFs for wildlife will require an analysis of the existing congener specific database. Within the last year the Risk Assessment Forum in U.S. EPA held a national workshop to address the development and use of TEFs for PCBs. The results of this workshop indicate that the use of PCB TEFs is problematic from a number of perspectives and the endorsement of specific TEFs, which can span 3 orders of magnitude for some PCB congeners, was not provided (U.S. EPA, 1991). To date the SAB has not addressed the PCB TEF issue for

wildlife, although it is my understanding that the topic is currently under consideration. Therefore, Dr. Nisbet's suggestion that the U.S. EPA has developed procedures for deriving wildlife criteria with TEFs is inaccurate.

The issue of chemical mixture toxicity is a critical area of research within ORD because there are not currently available sufficiently developed methods to quantify long term risks. As I indicated previously, ORD is currently reassessing the risks of dioxin, and assuming sufficient funding, plans to develop defensible TEFs. Because, in my opinion, there are currently inadequate means to assess risks of chemical mixtures to wildlife, I believe that it is rational to proceed with defensible chemical-specific assessments to reduce the risks of chemical pollutants to wildlife. I would certainly support the development of specific criteria and TMDLs for PCBs, DDE, etc. to further protect wildlife in the Columbia River Basin; however, the matter at hand is specific to dioxin.

Paragraph 11. The SAB reviewed the Great Lakes Water Quality Initiative (GLWQI) in mid-February and a preliminary oral summary of their findings did not indicate any major criticisms with the approach. A written report will probably not be available for several months. Please note that Dr. Nisbet served on this committee as a consultant.

Paragraph 14. Ph.D. dissertations are used from time to time in generating aquatic life criteria. Ph.D. dissertations can be readily obtained through common literature retrieval services for review. In any event, I have been in contact with the authors of the manuscript describing the study on which the NOAEL is based, and I have been informed that it will be published in the March issue of the Journal of Toxicology and Environmental Health. The manuscript describing a series of egg injection studies has been submitted to Environmental Toxicology and Chemistry (see comments on paragraph 16, below).

Note that Dr. Nisbet does not challenge the quality of the Nosek (1991) study, he only indicates it needs review. I can only speculate that he does not challenge the quality of the study because it was done under the direction Dr. Richard Peterson at the University of Wisconsin-Madison, who is considered a leading researcher in this area of investigation. Although Dr. Nisbet feels the exposure regime in the cited study raises questions, he fails to specify what these questions may be. In my declaration I clearly state the assumptions associated with the exposure regime; however, he does not address my analysis in this regard so I am at a loss as to how to respond to his open-ended statement.

Paragraph 15. Dr. Nisbet and I have a reasonable difference of opinion on whether or not an uncertainty factor of 3 or 6, for extrapolating the toxicity of PCBs between pheasants and chickens, would be appropriate. The uncertainty factor of 3 is consistent with U.S. EPA guidance (U.S. EPA, 1990). The critical point is that I used an interspecies uncertainty factor of 10 in the eagle dioxin assessment, which is more than adequate even if one accepts Dr. Nisbet's factor of 6.

Paragraph 16. Dr. Nisbet's suggestion of using a 50-fold interspecies uncertainty factor, in my opinion, is not proper. The factor he proposes in this Paragraph is based on a comparison of embryo toxicity values from egg injection studies using a PCB congener. The egg injection exposure route is not comparable to the pheasant study used in my analysis because in the pheasant study dioxin was administered to the hens and effects to embryos (mortality, hatchability, etc.) are due to exposure from dioxin depuration in the egg. The hen exposure scenario is appropriate for consideration of dioxin effects to the bald eagles in the Columbia river basin for the reasons described in the next paragraph. Even if one accepts egg injection studies as the basis for establishing interspecies uncertainty factors when females are exposed, which I do not, better data is available where dioxin, instead of a PCB congener, is directly studied. Based on egg injections, Nosek (1991) calculated a dioxin LD50 for embryo mortality of 1,300 pg/g egg for pheasants, while Allred and Strange (1977) reported a dioxin LD50 of 240 pg/g egg in chickens. These data indicate that the chicken embryo is approximately 5 times more sensitive. Again the 10-fold interspecies uncertainty factor I used in the eagle dioxin risk assessment seems more than reasonable. Please note that the use of the interspecies uncertainty factor of 10 assumes that the eagle is more sensitive than the chicken. In fact, based on studies to date, birds like the chicken, pheasant, and bobwhite quail are probably the most sensitive species (e.g., see Grieg et al., 1973; Hudson et al., 1984; Nosek, 1991) and therefore the 10-fold uncertainty factor is certainly conservative.

Paragraph 17. The study by Cheung et al. (1981) is difficult to use for two reasons. First, Cheung et al. addressed histopathological endpoints in chickens and did not use doses high enough to establish NOAELS for embryo mortality, hatchability, etc. Therefore this study is of questionable utility. In my opinion, histopathological endpoints in developing embryos should be evaluated in relation to the health of individuals and the viability of a population. At what incidence rate for histopathological alterations in embryos would one predict significant changes in the dynamics of a bird population? I am not able to make such a projection. Dr. Nisbet does not offer any insights either and I can only assume he does not wish to provide such speculation. The second problem is

associated with egg injection studies, in general, and relates to the exposure assessment. In my opinion, there is great uncertainty in relating aqueous dioxin concentrations to eagle egg concentrations at this time. Intensive site-specific BAF values for avian eggs are needed before site-specific risk assessments can be performed with confidence and more research on the toxicokinetics and bioavailability of superhydrophobic chemicals is required to relate female dioxin concentrations to egg residue levels. Therefore, in my opinion, the use of reproduction studies that involve exposures to the females are more defensible for the dioxin assessment because: 1) possible effects on the female physiology (e.g., reproductive hormones) as well as direct effects on the embryo are integrated and 2) exposure of the egg, through depuration from the female, is more reflective of actual environmental exposure.

Paragraphs 18, 19. Dr. Nisbet again cites prey identification studies to claim extremely large proportions of birds in the eagle diet. He again ignores Garrett et al.'s (1988) own conclusion that these estimates are biased and that a value of 90% to 94% fish in an eagle diet is more reasonable. As I indicated in my declaration a study by Kozie and Anderson (1991) suggests that fish comprise about 97% of an eagle diet and that other workers have indicated that if available, fish can make up 100% of the diet. If it is assumed that eagles eat 3%, 6% or 10% birds in their diet, the impact on my assessment, which assumes a 100% fish diet, is not significant. For the sake of argument assume that a dioxin BAF for a fish eating bird is 30 times higher than a dioxin BAF for a fish (a value offered by Dr. Nisbet). Next, based on the work of Stalmaster and Gessaman (1982), it can be estimated that eagles would consume about 0.25 kg bird/day, using the mallard as a model food source, to meet their energy requirements. In the original assessment it was estimated that eagles eat 0.5 kg fish/day to meet their energy requirements. There is more energy in birds (2.0 kcal/g) than fish (1.0 kcal/g), therefore eagles would eat fewer kg of birds than fish to achieve the needed energy intake of 500 kcal/day (Stalmaster and Gessaman, 1982). Finally, Garrett et al. (1988) document in Table 2.1 of their report that of the birds consumed by eagles, approximately 40% are non-fish eating (e.g., the mallard). These birds would not bioaccumulate dioxin to the extent that fish eating birds would and in fact they may have less dioxin than would fish. Assuming that non-fish eating birds have dioxin levels comparable to fish, which is a conservative assumption (their food sources, which include vegetation, should contain TCDD at levels below that observed in fish at the top of an aquatic food chain), the dioxin exposure to eagles with 3%, 6%, or 10% birds in the diet would be about 1.2, 1.5, or 1.8 times higher than the exposure associated with a 100% fish diet. Although these exposures would lead to dioxin intakes over the 140 pg dioxin/kg bald eagle/day no effect level calculated in my

earlier declaration, I consider the differences in exposure insignificant in light of the conservative approaches used to address uncertainties in the overall analysis.

Dr. Nisbet's claim that 3% birds in the diet would contribute as much dioxin as the fish in the remaining diet (i.e., a 2-fold increase in exposure) is not consistent with my analysis; even with 10% birds in the diet the dioxin intake is not twice that of a 100% fish diet. I believe Dr. Nisbet is assuming that eagle intake of birds and fish are the same on a kg/day basis and that all the birds consumed are piscivorous, which is not correct. Since Dr. Nisbet did not provide any calculations to support his assertion it is impossible to determine how his conclusions were derived.

As I discussed in my declaration, there are uncertainties in assessing risk. While a risk assessment of the type I provided in my declaration generates a single numeric no adverse effect level, it would be incorrect to accord the precise value of the overall calculation undue significance given the range and types of uncertainties involved in the analysis. In this regard, measurement uncertainties refer to the usual variances that accompany scientific measurements and reflect the accumulated variances associated with individual values in the assessment. A different type of uncertainty is associated with assumptions that must be made in the face of data gaps (e.g., interspecies differences in response to chemicals). Given the conservative nature of the uncertainty factors used to account for data gaps and the variability associated with eagle exposure parameters in the dioxin risk assessment, I contend that: 1) there is no significant difference in the calculated dioxin risk for the varying dioxin intakes associated with 0 to 10% birds in a bald eagle diet and 2) that it is reasonable to assume that an ambient aqueous dioxin concentration of 0.013 ppq will be protective for eagles, even though there is variability in the actual hazards and exposures.

Paragraph 20. The issues discussed above rebut Dr. Nisbet's overall conclusions.

Paragraphs 21, 25e. Dr. Nisbet is now providing a more forthright discussion and acknowledging that dioxin levels would eventually be reduced in the Columbia River basin. As a point of reference, research in our laboratory indicates a significant decline in dioxin levels in Lake Ontario with a reduction in loading. I am pleased that Dr. Nisbet agreed with my declaration that a modeling exercise is needed to quantify dioxin levels associated with the TMDL. Dr. Nisbet, however, still does not provide a calculation to support his qualitative claim that suggests a 95% reduction in dioxin loading from pulp and paper mills would result in significantly elevated dioxin levels,

compared to an implied 100% reduction in dioxin loading. I feel the discussion Dr. Nisbet provides in Paragraph 21 is quite weak in comparison to assertions in his original declaration.

Please feel free to call or write if you have any questions on this matter.

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